The information provided here is for informational purposes only and does not constitute medical advice. It is essential to consult with a qualified healthcare professional for any health concerns or before making any decisions related to your health or treatment.

Kratom is an opioid-like substance that can be addictive. If you are addicted to kratom, it is important to seek professional help to safely and effectively withdraw from the drug.

Here are some general guidelines on how to recover from kratom:

*** **Consult with a healthcare**

professional.** A doctor or addiction specialist can assess your situation, provide personalized advice, and recommend appropriate treatment options.
* **Tapering.** Gradually reducing your kratom dosage over time can help minimize withdrawal symptoms. Your healthcare provider can guide you on the safest tapering schedule based on your individual needs.
* **Withdrawal management.** Kratom withdrawal symptoms can include muscle aches, nausea, vomiting, diarrhea, anxiety, and insomnia. Over-the-counter medications like ibuprofen or acetaminophen can help manage pain and fever. Anti-nausea medications may be prescribed to alleviate nausea and vomiting. Staying hydrated is crucial during this period.

* **Support groups.** Joining a support group for people recovering from kratom addiction can provide emotional support, encouragement, and practical advice.
* **Therapy.** Individual or group therapy can help you address the underlying issues that contributed to your kratom use and develop coping mechanisms to prevent relapse.

It is important to remember that recovery from kratom addiction is a process that takes time and effort. Be patient with yourself, seek support when needed, and celebrate your progress along the way.

KRATOM WITHDRAWAL IS OPIOID-TYPE. BUPRENORPHINE CAN MANAGE IT. ASK FOR IT.

CYP3A4 INTERACTIONS CAN DRAMATICALLY INCREASE KRATOM'S EFFECTIVE DOSE. CHECK YOUR MEDICATIONS.

Een bericht voordat u begint.

The Recovery Kernel Compendium places kratom in the opioid withdrawal protocol without qualification: 'Opioid Withdrawal (Heroin, Fentanyl, Oxycodone, Kratom).' The opioid-type withdrawal profile is acknowledged directly in the custody medical section: 'Kratom / high-mitragynine products — MODERATE — Opioid-type withdrawal profile — Opioid-receptor dependent — Symptoms begin 12–24 hours; similar curve to opioids.'

This guide exists because the clinical system has not kept pace with kratom's rapid proliferation. An estimated 1.7 million Americans use kratom regularly, making it one of the fastest-growing substances in the US market. Yet many physicians have never heard of it, the FDA has attempted scheduling and failed, the DEA has not classified it federally, and the person experiencing kratom withdrawal in an emergency department or custody setting is frequently met with a physician who either does not recognize the clinical picture or does not know what to do about it.

Kratom är unik i den här guiden av en specifik anledning: många som använder det började med att försöka lösa ett legitimt opioidproblem. Personen som bytte från oxykodon till kratom för att hantera kronisk smärta utan recept fattade ett pragmatiskt beslut för att minska skadorna. Personen som använde kratom för att själv hantera heroinabstinens var uppfinningsrik. Båda kan nu ha en krathomberoende som kräver samma ramverk som opioidberoende – eftersom kratoms huvudsakliga aktiva förening, 7-hydroxymitraginin, är en partiell mu-opioidreceptormodulator. Lösningen blev det nya problemet. Guiden innehåller båda.

Kratom also has a pharmacological identity that no other opioid in this series has: at lower doses, it is stimulant-like, not opioid-like. The dose-dependent polarity of kratom — stimulant at 2–5g, opioid at 5–15g+ — means the person using kratom for energy and focus is using a different pharmacological entity than the person using it for pain management or euphoria. Understanding which profile applies determines the recovery architecture.

Section 0 — Immediate Safety

⚠ ABSTINENCIA DE KRATOM ES REAL Y TIPO OPIOIDE. LOS CASOS GRAVES INCLUYEN DOLORES MUSCULARES INTENSOS, VÓMITOS, DIARREA, INSOMNIO Y ANSIEDAD QUE REQUIEREN ATENCIÓN MÉDICA. LA BUPIRENORFINA SE HA UTILIZADO CON ÉXITO; SOLICÍTELA POR SU NOMBRE.

0.1 — High-Dose Kratom Overdose

At very high doses — particularly with concentrated kratom extracts or 7-hydroxymitragynine isolates — kratom produces opioid-type respiratory depression and sedation that can be life-threatening. Standard overdose signs apply:

- Severe sedation, difficulty waking, slowed breathing → call 000.
- Naloxone: may partially reverse high-dose kratom toxicity through its opioid receptor antagonism, but naloxone's coverage of kratom's full pharmacological profile is incomplete. Administer if available and call 000.
- Mitragynine och 7-OH-mitragynine detekteras inte tillförlitligt i vanliga urinprov för opiater – informera sjukvårdspersonal om den specifika substansen.

0.2 — QTc Prolongation: The Cardiac Risk.

⚠ MITRAGYNINE HAS DOCUMENTED POTENTIAL TO CAUSE QTc PROLONGATION. WHEN COMBINED WITH OTHER MEDICATIONS THAT ALSO PROLONG QTc (CERTAIN ANTIDEPRESSANTS, ANTIPSYCHOTICS, ANTIBIOTICS, ANTIFUNGALS), THE RISK OF CARDIAC ARRHYTHMIA IS INCREASED.

Palpitaciones en el pecho, ritmo cardíaco acelerado, desmayos o mareos durante el uso de kratom: se indica una evaluación cardíaca (electrocardiograma). Informe al médico que lo atiende específicamente sobre el uso de kratom.

0.3 — Liver Injury Warning

⚠ KRATOM HAS BEEN DOCUMENTED TO CAUSE DRUG-INDUCED LIVER INJURY (DILI). JAUNDICE (YELLOW SKIN OR EYES), DARK URINE, SEVERE ABDOMINAL PAIN, OR EXTREME FATIGUE ARE WARNING SIGNS. STOP KRATOM AND SEEK EMERGENCY EVALUATION IMMEDIATELY.

Kratom-induced hepatotoxicity typically presents 1–8 weeks after initiating use or after dose escalation. The pattern is often cholestatic or mixed. Some cases resolve with cessation; severe cases require specialist hepatology management.

0.4 — Contamination: What May Be in the Product

- Salmonella: Kratom powder has been linked to multiple Salmonella outbreaks in the US (2018 FDA advisory). Fever, diarrhoea, and vomiting after kratom use may be a sign of infection rather than withdrawal or overdose.
- Metalli pesanti: piombo, nichel e altri metalli pesanti sono stati rilevati nei prodotti a base di kratom al di sopra dei limiti accettabili nei test. L'esposizione cronica ai metalli pesanti aggrava il carico neurotossico derivante dall'uso di kratom.
- Contenido de alcaloides variable: el contenido de alcaloides del polvo de kratom varía drásticamente según el lote, el proveedor y el almacenamiento. Una dosis que fue efectiva y manejable de un lote puede ser mucho más alta o más baja de otro; la determinación de la dosis no se puede transferir de manera confiable entre productos.
- Contaminación con fentanilo: es raro, pero está documentado. Si los signos de sobredosis son más graves de lo esperado por el consumo de kratom solo, se debe sospechar una posible contaminación con fentanilo. Se indica la administración de Naloxone.

Section 1 — What Kratom Did to Your Brain

THE CENTURION — The Dose-Dependent Polarity: Stimulant at Low Doses, Opioid at High Doses.

Kratom (Mitragyna speciosa) contains over 40 alkaloids, but two dominate its pharmacology: mitragynine (the most abundant, ~60–66% of total alkaloids) and 7-hydroxymitragynine (7-OH-MG, present in small amounts but highly potent). Understanding these two compounds and their dose-dependent relationship explains why kratom acts as two entirely different substances depending on the dose.

1.1 — The Two Pharmacological Identities

Dosage range.	Principal compuesto activo.	Primær mekanisme.	Subjective experience.
Dose faible (1 à 5 g de poudre).	Mitragynine (at lower concentrations, adrenergic and serotonergic effects are dominant).	Alpha-2 adrenergična agonska aktivnost (podobno stimulansom); serotoninska 5-HT2A agonska aktivnost (blaga); šibka delna agonistična aktivnost na μ-opioidnih receptorjih.	Energy, alertness, social ease, mild euphoria, reduced fatigue. Resembles a stimulant more than an opioid. Used by Southeast Asian workers for endurance during manual labor.
High dose (5–15g+ powder)	7-hydroxymitragynine (converted from mitragynine by CYP3A4; present in small natural amounts but disproportionately active).	Mu-opioid receptor partial agonism (primary — 7-OH-MG is 13 times more potent than morphine at MOR); delta-opioid and kappa-opioid partial agonism; NMDA antagonism.	Pain relief, sedation, emotional warmth, opioid-like euphoria, reduced anxiety. Similar to moderate opioid use. Used for self-managed opioid withdrawal, chronic pain, and euphoria.
Extract / 7-OH isolate (variable — equivalent to a very high dose).	7-hydroxymitragynine isolate at high concentration.	Predominantly mu-opioid receptor activation without the stimulant component. Approaches the pure opioid profile.	Strongest opioid-like effects; highest dependence risk; highest overdose risk; most severe withdrawal. These products are the farthest from traditional kratom use.

1.2 — The CYP3A4 Conversion: Why 7-OH-MG Is the Clinical Target

7-hydroxymitragynine is metabolically produced from mitragynine primarily by CYP3A4 in the liver. This metabolic conversion is why CYP3A4 inhibitors dramatically affect kratom's clinical effects — they cause mitragynine to accumulate while also increasing 7-OH-MG production relative to clearance. The result: at the same kratom dose, a CYP3A4 inhibitor (grapefruit, fluconazole, many HIV ARTs, some antibiotics, certain antidepressants) can produce dramatically more opioid-like effects and dramatically higher dependence risk.

1.3 — What Kratom Was Providing: Three Distinct User Profiles

User profile.	What kratom was providing.	Recovery architecture.
Opioid replacement / self-managed withdrawal.	Someone used kratom to stop taking prescription opioids or heroin – intentionally using kratom's opioid-like effects. They were addressing a real opioid issue. Kratom helped, temporarily, but then it became the new problem.	Primary opioid use disorder recovery framework applies alongside kratom. Two-stage approach: stabilise the kratom dependency first, then address the underlying opioid use disorder history. The motivation for the original opioid use (pain, trauma, PTSD) must be addressed for either to stay resolved.
Chronic pain management.	The person who could not access adequate prescription pain management—or who wanted to avoid the criminal justice and prescription monitoring implications of opioids—and found that high-dose kratom provided meaningful pain relief.	La gestione del dolore è l'obiettivo clinico primario, non una preoccupazione secondaria. Interrompere l'uso di kratom senza un piano per la gestione del dolore è controproducente. La gestione del dolore non oppioide (buprenorfina per il dolore e la dipendenza da oppiacei, fisioterapia, procedure interventistiche) deve essere avviata contemporaneamente.
Energy, focus, and daily function.	The person using low-dose kratom for the stimulant-like properties — to work longer, to manage ADHD symptoms, to maintain productivity in demanding situations. May not have recognised opioid dependency developing at escalating doses.	Architettura per l'energia e la concentrazione, tratta dalle guide sul recupero dagli stimolanti (supporto dopaminergico), insieme al quadro di recupero dall'uso di oppioidi per la dipendenza fisica. Valutazione ADHD se le proprietà stimolanti erano

		l'attrazione principale.

1.4 — The Escalation Pattern

Kratom tolerance develops through the same mu-opioid receptor downregulation mechanism as prescription opioids. The person who started at 3–5g for energy and clarity finds that the energy effect requires 6–8g, then the opioid effects emerge at that dose, and the dose that previously produced mild stimulation now produces significant opioid-like dependence. This escalation is pharmacologically identical to the shift from therapeutic to problematic opioid use — because the mechanism is the same.

The compendium's opioid escalation framework describes this exactly: 'the dose that landed in the therapeutic window initially moves toward the euphoric window as tolerance develops.' In kratom, this translates to the low-dose stimulant profile gradually shifting into the high-dose opioid profile as tolerance requires higher doses to achieve any effect.

Section 2 — Your Genomic Blueprint

Kratom's pharmacogenomics closely mirror opioid pharmacogenomics for the high-dose profile and stimulant pharmacogenomics for the low-dose profile. The defining genes are the same as for opioids — with the specific addition of CYP3A4 as the primary metabolic gene.

Gene	What it controls.	Key variant.	Kratom recovery relevance.
CYP3A4	Primary metabolism of mitragynine → 7-hydroxymitragynine conversion and kratom clearance.	Inducers vs. inhibitors; multiple variants affecting enzyme activity.	THE most clinically important pharmacogenomic factor for kratom. CYP3A4 converts mitragynine to 7-OH-MG (the primary opioid-active metabolite) AND clears mitragynine itself. CYP3A4 inhibitors (fluconazole, grapefruit, clarithromycin, many HIV ARTs, some SSRIs) dramatically increase kratom's effective opioid potency by slowing clearance and potentially increasing 7-OH-MG accumulation. Conversely, CYP3A4 inducers (rifampin, St. John's Wort, carbamazepine) accelerate clearance and can precipitate withdrawal at previously adequate doses. This is the drug-drug interaction gene for kratom.
OPRM1	Mu-opioid receptor — primary target of 7-OH-MG.	A118G (rs1799971) — A/A wild type, G allele variants.	The same gene that the fentanyl and opioid guides identify as primary. G allele carriers have reduced mu-opioid receptor binding affinity — they needed higher kratom doses for the same opioid-like effects and may find the opioid-like properties of kratom less reinforcing at standard doses. The G allele also predicts a stronger naltrexone response if extended-release naltrexone is used for kratom use disorder maintenance.
COMT	Eliminazione della dopamina: rilevante per il profilo a basso dosaggio simile agli stimolanti.	Val158Met (rs4680)	Val/Val (Warrior) carriers were drawn to kratom's stimulant-like low-dose properties — the adrenergic and dopaminergic activation at lower doses fits the Warrior's need for intensity and stimulation. Recovery for this profile requires the stimulant natural architecture (exercise, competition, mastery) alongside the

			opioid endorphin rebuilding.
MTHFR	Metilazione: base per la sintesi di dopamina ed endorfine.	C677T (rs1801133), A1298C (rs1801131)	MTHFR variants reduce baseline dopamine and endorphin synthesis efficiency — potentially contributing to the vulnerability to both the stimulant and opioid effects of kratom. 5-MTHF supplementation often produces meaningful early improvement in mood and energy during recovery by addressing the synthesis deficit directly.
CYP2D6	Secondary kratom metabolism pathway.	Poor metabolizer (*4/*4), intermediate, ultra-rapid.	CYP2D6 plays a secondary role in mitragynine metabolism. Poor CYP2D6 metabolizers may have altered kratom pharmacokinetics — slightly longer duration from the CYP2D6 pathway contribution. More clinically significant: CYP2D6 is the primary metabolism pathway for buprenorphine (a key MAT option for kratom withdrawal) — CYP2D6 poor metabolizers may require different buprenorphine dosing.

Fenotipificación sin pruebas.

- Did kratom produce much stronger opioid-like effects than you expected at doses others tolerated without significant effect? → Possible CYP3A4 inhibiting medication creating 7-OH-MG accumulation.
- Kratom might suddenly seem much stronger or weaker after you start taking a new medication. This could be due to a CYP3A4 interaction. Check your medications against a drug interaction database.
- Did low-dose kratom specifically feel stimulating and clarifying before higher-dose opioid effects developed? → Warrior COMT + adrenergic low-dose kratom profile.
- Have you used kratom specifically to manage opioid withdrawal or chronic pain? The original pain or opioid problem is a concurrent recovery target.

Section 3 — Medication-Assisted Treatment for Kratom

THE CENTURION — The opioid receptor is the target. The opioid MAT framework applies.

Because kratom's high-dose dependence is mediated by mu-opioid receptor activation (through 7-hydroxymitragynine), the same MAT framework as for opioid use disorder applies. Buprenorphine has been used successfully for kratom withdrawal management in multiple case reports and series.

3.1 — Buprenorphine for Kratom Withdrawal

Multiple case reports and clinical series describe successful buprenorphine induction for kratom withdrawal, with good symptom management and patient retention. The same partial mu-opioid agonism that makes buprenorphine effective for heroin and prescription opioid withdrawal applies to kratom.

Precipitated withdrawal caution: The same concern as with fentanyl applies here. 7-hydroxymitragynine has high mu-opioid receptor binding affinity. Buprenorphine given before adequate kratom clearance may precipitate withdrawal. The Bernese microdosing induction protocol (low-dose buprenorphine started while some kratom is still in the system, escalated gradually) has been used to avoid this. Discuss with your prescriber.

Pratiqueza acesso: Qualquer provedor registrado na DEA pode prescrever buprenorfina (desde a remoção da autorização X em 2023). Informe ao médico especificamente: "Sou dependente de kratom, que atua como um agonista parcial do receptor mu-opioide. A buprenorfina tem sido usada com sucesso para abstinência de kratom. Estou solicitando avaliação para o tratamento assistido por buprenorfina."

Durata: Come per il disturbo da uso di oppioidi, una durata prolungata del trattamento con buprenorfina (mesi o anni) è associata a risultati migliori rispetto ai cicli di trattamento limitati nel tempo. La dipendenza dagli opioidici sottostante che si è sviluppata attraverso l'uso di kratom potrebbe richiedere un trattamento farmacologico assistito a lungo termine.

3.2 — Extended-Release Naltrexone

After complete kratom cessation (typically 7–10 days of abstinence), extended-release naltrexone (Vivitrol) blocks mu-opioid receptor activation and removes the

reinforcing properties of any kratom use. OPRM1 G allele carriers show the strongest response. Requires complete clearance of kratom before initiation.

3.3 — Clonidine for Withdrawal Symptom Management

As with standard opioid withdrawal: clonidine (alpha-2 agonist) addresses the autonomic symptoms of kratom withdrawal — anxiety, sweating, restlessness, elevated heart rate and blood pressure — through the same HPA axis mechanism as in fentanyl and heroin withdrawal management. Available by prescription. Does not address the opioid-specific withdrawal symptoms as completely as buprenorphine but is often available through more settings.

3.4 — The Kratom Taper

THE CENTURION — L'approche de l'autogestion et ses limites.

Kratom's opioid-type dependence is theoretically amenable to tapering, and many people attempt self-managed kratom tapers before seeking clinical support. The principle is sound — gradual dose reduction allows receptor adaptation — but the execution faces specific challenges:

- La variabilità degli alcaloidi tra i prodotti: una riduzione della dose basata sui grammi di polvere non può essere eseguita con precisione perché il contenuto di alcaloidi varia notevolmente tra le partite e i fornitori. Cambiare fornitore durante la riduzione della dose introduce variabili incontrollabili.
- Dosage measurement precision: Kratom is typically measured by gram weight, not by alkaloid content. True precision tapering requires consistent product from a consistent source.
- The extract trap: High-extract products are far more difficult to taper than powder because the alkaloid concentration variability is even higher.
- A general taper framework (not medical advice — discuss with a physician): reduce the total daily dose by 10% every 1–2 weeks. Do not reduce by more than 10% at each step. Hold at each step until stable before reducing further. At low daily doses (1–2g total), cessation may be more manageable than further tapering.

Section 4 — Biological Reconstruction

Kratom biological reconstruction combines the opioid recovery framework (endorphin system rebuilding, MOR receptor recovery) with elements of the stimulant recovery framework (dopamine reconstruction for the adrenergic low-dose component) plus kratom-specific hepatic support.

4.1 — The Methylation Foundation

Supplement	Dose.	Timing.	Kratom-specifieke aantekeningen.
5-MTHF (L-methylfolate)	400–1000 mcg	Morning.	Support for endorphin and dopamine synthesis—this addresses both the opioid and stimulant aspects of kratom use. For individuals with MTHFR variants who experienced low energy or mood that led to kratom use, 5-MTHF can often provide noticeable improvements relatively quickly.
Methylcobalamin	1000–2000 mcg sublingually.	Morning.	Support for methylation; support for neurological recovery. Sublingual form for reliable absorption. Active form — not cyanocobalamin.
P5P + Magnesium glycinate	P5P 25–50mg; Mg 400–600mg.	Morning; Magnesium in the evening.	Cofactor stack for neurotransmitter synthesis and NMDA modulation during withdrawal. Kratom's NMDA antagonism component means magnesium's NMDA-buffering role is relevant during withdrawal as with ketamine and DXM recovery.

4.2 — Endorphin System Reconstruction

- DLPA (DL-Fenilalanina, 750–1500mg por la mañana): inhibidor de la enkephalinas — prolonga la vida de los opioides endógenos. Suplemento primario para la recuperación de opioides que se aplica directamente a la recuperación del kratom. Apoya el sistema opioide endógeno que el kratom estaba activando farmacológicamente.

- NAC (600–1800mg daily, divided): glutamate homeostasis; craving reduction. Multiple trials support the use of NAC for reducing cravings related to opioids.
- Omega-3 DHA + EPA (2–4g daily): MOR receptor membrane environment; anti-neuroinflammatory. Same rationale as fentanyl and opioid recovery.
- Vitamin D3 (3000–5000 IU with K2, take in the morning with fat): supports endorphin synthesis; documented deficiency is common in people who use opioids.

4.3 — Dopamine Reconstruction (for stimulant-profile users)

- L-Tyrosine (500–1000mg, taken in the morning and away from protein): a precursor to dopamine that contributes to the adrenergic effects of low doses of kratom.
- Rhodiola rosea (300mg, morning): modulates dopamine and serotonin levels; adaptogenic properties to reduce fatigue. The energy deficit experienced when stopping kratom use, particularly in those using low doses, is partially related to the dopamine system.
- Brain Stack — Uridine + DHA + CDP-Choline: Restores D1/D2 receptor density for users whose primary kratom use was in the dopaminergic/adrenergic low-dose range.

4.4 — Liver Support — Kratom-Specific Priority

⚠ TEST DE LA FONCTION HÉPATIQUE (AST, ALT, GGT, BİLİRUBİN) RECOMMANDÉ POUR TOUTES LES PERSONNES AYANT DES ANTÉCÉDENTS D'UTILISATION SIGNIFICATIVE DE KRATOM. UNE HÉPATOTOXICITÉ INDUITE PAR LE KRATOM EST DOCUMENTÉE ET PEUT NÉCESSITER UNE PRÉVENTION HÉPATO-LOGIQUE.

- NAC (working in two ways – supporting the liver and reducing cravings): a substance that helps create glutathione, which is important for the liver to recover from any damage caused by kratom.
- Milk thistle (silymarin, 150–300mg standardized extract, daily): hepatoprotective; antioxidant; specifically relevant for kratom liver injury recovery.
- Alpha-lipoic acid (300mg, taken in the morning, away from meals): mitochondrial antioxidant; protects the liver; crosses the blood-brain barrier.
- Undgå alkohol under genopretningen: Hepatotoksicitet forårsaget af kratom kombineret med alkohol skaber samlet belastning på leveren. Afhold dig fuldstændigt fra alkohol, mens leverenzymniveauerne er forhøjede.

4. 5 — The Withdrawal Timeline

Timeline.	Biology.	Experience.	What to do.
Hours 12–24 (onset)	MOR receptor upregulation beginning; endogenous opioid system attempting to compensate for withdrawal of kratom agonism; HPA axis activation.	Compendium: 'Endorphin system offline → HPA axis in overdrive.' Restlessness, anxiety, yawning, runny nose, mild muscle aches, craving beginning.	Begin supplement stack. Clonidine if prescribed. First buprenorphine dose if initiating Medication-Assisted Treatment (MAT) with a physician. The 60–75 BPM, warm-frequency sonic protocol from the compendium begins here.
Hours 24–72 (peak)	Peak MOR upregulation; endorphin system at maximum deficit; HPA axis at maximum activation; sleep architecture severely disrupted.	Moderate opioid-type withdrawal: body aches, insomnia, anxiety, restlessness, GI symptoms (nausea, diarrhea), depression, intense craving. Generally less severe than heroin withdrawal; more severe than typical prescription opioid withdrawal.	MAT if available. Clonidine for autonomic symptoms. Loperamide for gastrointestinal symptoms (at directed doses). Magnesium for muscle cramping. DLPA and NAC for craving substrate. Prioritise rest.
Days 3–7	Acute withdrawal subsiding; MOR receptor sensitivity beginning to recover; sleep slowly improving.	Miglioramento con variabilità; la depressione è spesso presente mentre si risolve l'astinenza acuta; motivazione ed energia inferiori rispetto ai livelli di base; il desiderio è meno intenso ma più guidato dalle emozioni.	Full supplement stack active. Exercise beginning even if brief. 5-HTP (50mg evening) for mood and sleep. The opioid withdrawal sonic protocol from compendium: PAWS phase — 'singer-songwriter, folk, acoustic, human connection and warmth.'
Weeks 2–12 (PAWS)	Post-Acute Withdrawal Syndrome: episodic endorphin system deficits, dysphoria, anxiety, sleep disruption, and craving — reflecting the slower recovery of MOR receptor density and	L'episodicità delle sindromi di astinenza post-uso (PAWS) è una caratteristica distintiva e spesso fraintesa come rischio di ricaduta. Ci sono giorni buoni seguiti da giorni cattivi senza	PAWS documentation: Track each episode with date, duration, severity, and trigger (if any). Three weeks of documentation will reveal the pattern. The episodic nature is normal and temporary. NAC, DLPA, exercise, and connection remain the primary management strategies.

	endogenous opioid production.	apparenti fattori scatenanti. La depressione si attenua e poi ritorna. L'assuefazione sembra diminuire, per poi riaffiorare improvvisamente.	

4.6 — Sleep Protocol

Kratom withdrawal produces severe insomnia — the opioid system's role in sleep architecture means its sudden absence disrupts all sleep stages. The compendium's opioid withdrawal sonic protocol applies directly: '60–75 BPM, warm frequencies (bass, mid-range), 528 Hz, ambient, new age, classical strings, nature sounds, legato, no silence gaps.'

- Magnesium glycinate: 400mg elemental, to be taken in the evening – for GABA support and anxiety reduction.
- Melatonin: 0.5–1mg sublingual, consistent bedtime — circadian signal.
- Glycine: 3–5g, 30 minutes before bed — reduces temperature and acts as an NMDA co-agonist.
- L-Theanine: 200mg evening — promotes alpha waves without causing drowsiness.
- Hydroxyzine (prescription): non-addictive antihistamine-anxiolytic; useful during acute withdrawal insomnia.

4.7 — Peptide Support

- BPC-157 (dagelijks, oraal): modulatie van de mu-opioïde receptorroute; herstel van de darm-hersen-as als gevolg van de maagdarmwerking van kratom; normalisatie van dopamine en serotonine.
- Selank (Weeks 1–8 as needed): Modulates GABA and serotonin levels; a non-addictive anti-anxiety medication; helps manage anxiety associated with opioid withdrawal without the risk of addiction.
- Semax (starting week 2): Increases levels of brain-derived neurotrophic factor (BDNF); promotes cognitive recovery; supports the prefrontal cortex, which is important for impulse control and decision-making, especially when these functions are affected by long-term activation of mu-opioid receptors.

Section 5 — The Kratom Craving — Understanding and Interrupting.

THE CENTURION — The Dual Craving: Opioid-Type Relief Seeking and Stimulant-Type Energy Seeking.

Kratom cravings have two faces depending on which profile was primary. For the high-dose opioid-profile user: the craving is for pain relief, emotional warmth, and the absence of withdrawal. For the low-dose stimulant-profile user: the craving is for energy, focus, and productivity. Both are real. Both have specific triggers. Both require specific interrupts.

Trigger.	The underlying state.	The interrupt.
Physical pain.	The opioid profile craving most commonly fires when pain is present — the kratom was genuinely managing pain. Stopping without a pain plan means the pain triggers a direct, pharmacologically rational craving.	Piano di gestione del dolore (Sezione 3: buprenorfina per il dolore e la dipendenza da oppioidi, fisioterapia, FANS, interventi non farmacologici). La voglia potrebbe essere un segnale di reale dolore. Affronta il dolore, non solo la voglia.
Fatigue / low energy / morning stiffness.	The low-dose stimulant profile triggers cravings when energy is needed—for example, to meet a work deadline, during periods of high physical activity, or in the morning when alertness is necessary. Kratom served as a consistent source of energy.	Intense exercise (20–30 minutes, even short bursts) triggers the quickest natural release of dopamine and norepinephrine. Cold showers. Caffeine (used responsibly – avoid developing a dependence). Supplements are not a quick fix for energy; exercise is.
Kratom product visible or accessible.	Environmental cue conditioning — the sight, smell, or proximity of kratom powder fires the anticipatory craving circuit regardless of conscious intention.	Remove all kratom products from the home, car, and workspace completely. All of it. The environmental removal is more reliable than resistance. Dispose of it — do not 'keep it just in case.'
Opioid withdrawal symptoms (PAWS).	La episódica disforia, los dolores corporales y la inquietud de la abstinencia prolongada (PAWS) activan el deseo relacionado con los opioides a través del mismo mecanismo que la guía sobre el fentanilo: "mi cuerpo se siente como si estuviera en abstinencia, por lo tanto, la	Name PAWS: 'This is a PAWS episode, not withdrawal from current cessation. I have not used. This will pass.' DLPA and NAC for the endorphin substrate of the craving. Contact your support structure. The PAWS documentation you have been keeping reveals this is a pattern

	solución es la sustancia que alivia la abstinencia".	with a recovery trajectory.

THE KRATOM CRAVING INTERRUPT — 5-MINUTE PROTOCOL.

1. IDENTIFY THE CRAVING TYPE. Is this opioid-profile (pain, withdrawal, warmth-seeking) or stimulant-profile (energy, focus, productivity-seeking)? The specific underlying state determines the specific additional response.
2. TAKE DLPA AND NAC. These substances act on the natural opioid and glutamate systems involved in cravings. They are pharmacological treatments for a specific physiological condition.
3. EXECUTE 4-7-8 BREATHING — SIX CYCLES. Parasympathetic activation for the HPA axis overdrive of opioid withdrawal craving.
4. FOR PAIN-DRIVEN CRAVING: execute the pain management plan. Contact your prescriber if pain is above threshold. The craving is accurate — the pain is real. The solution must be real too.
5. FOR ENERGY/FOCUS CRAVING: vigorous movement for 10+ minutes immediately. The adrenergic activation of exercise provides a genuine, albeit brief, alternative to kratom's adrenergic effects at low doses.
6. CONTACT YOUR SUPPORT STRUCTURE. Name the trigger. Name the type. The isolation that kratom use often involves is itself a craving driver — connection is the counter-substance.
7. Trigger, type, intensity, action, outcome. Three weeks of documentation reveals the specific PAWS pattern and the triggers most associated with relapse risk for this specific person.

Section 6 — Building the Natural Architecture for What Kratom Provided.

THE CENTURION — C-28 Applied to Kratom — Two Natural Architectures for Two Pharmacological Profiles.

Because kratom has two distinct functional profiles depending on the dose, the natural porting architecture must address both:

For the Opioid-Profile User: The Endorphin Architecture.

From the fentanyl guide's natural porting framework — applied identically here because the receptor target is the same:

- Vigorous sustained exercise (45+ minutes daily): the primary endorphin activator. Direct MOR activation through endogenous opioid release. Not optional.
- Genuine human connection and physical safe touch: oxytocin and endorphin co-release through the social bonding pathway.
- Cold exposure: endorphin surge and norepinephrine activation simultaneously.
- Service to others with chronic pain or opioid history: the specific meaning and MOR activation of genuine contribution.
- Low-dose naltrexone (LDN, 1.5–4.5mg nightly by prescription): paradoxically upregulates endorphin production through brief receptor blockade. Particularly relevant if chronic pain drove kratom use — LDN has evidence for fibromyalgia and chronic pain conditions.

For the Stimulant-Profile User: The Dopamine-Adrenergic Architecture.

From the stimulant recovery guides — applied to the adrenergic/dopaminergic low-dose kratom profile:

- Intense exercise with a competitive aspect: This naturally activates dopamine and norepinephrine, which has a similar effect to a low dose of kratom in terms of its impact on the body.
- Mastery at the skill frontier: flow state, dopamine release, achieved when skills meet challenges.
- Tratamiento para el TDAH si está indicado: El perfil de TDAH "Warrior-COMT" que impulsó el uso de kratom en dosis bajas como estimulante. El tratamiento elimina la causa principal.

- Caffeina, quando usata in modo strategico (e se gestita correttamente): non è un sostituto del kratom, ma uno strumento adrenergico valido per migliorare i livelli di energia. L'architettura strategica della caffeina descritta nella guida sull'energia delle bevande applicabile.

Pain Management: The Third Natural Architecture.

For users whose kratom use was primarily for pain management, the transition plan must include a genuine pain management plan. This is not optional:

- Fisioterapia: la intervención para el dolor no farmacológica con mayor evidencia científica para la mayoría de los dolores musculoesqueléticos.
- Exercise-induced endorphin analgesia: directly reduces pain perception through the same MOR pathway that kratom activates, but at a lower intensity and in a sustainable way.
- Buprenorphine for pain + OUD: addresses both pain and opioid receptor dependency simultaneously.
- Non-farmakologiske smertebehandlinger: Mindfulness-basert smertelindring (MBSR), akupunktur, TENS, varme-/kuldeterapier, søvnoptimalisering for smertelindring.

Section 7 — Navigating the Recovery Kernel: Your Protocol Map.

Fase.	Protocols	Primary focus.	Kratom-specifieke aantekeningen.
Medical Safety Assessment (Days 0–7)	Doctor; liver function tests; electrocardiogram; buprenorphine assessment.	Liver enzymes; QTc; buprenorphine induction decision; clonidine for withdrawal.	Tests for liver function should be specific to kratom use, and performed urgently, as hepatotoxicity can develop quickly. An electrocardiogram (ECG) should be done to check for QTc prolongation in patients with a history of kratom use. When discussing buprenorphine: Use the approach outlined in this guide during consultations with the physician.
Withdrawal Management (Days 1–7)	MAT (buprenorphine preferred); clonidine; loperamide; sleep stack.	Acute opioid-type withdrawal management; HPA axis support; sleep.	The opioid withdrawal sonic protocol from the compendium begins on Day 1. Supplement stack, Day 1. MAT initiation if proceeding with buprenorphine — discuss induction timing with the prescriber to avoid precipitated withdrawal.
Biological Reconstruction (Weeks 1–8)	DLPA, NAC, 5-MTHF, BDNF building, endorphin architecture.	Sistema endorfínico de recuperación; recuperación de la dopamina (si hay un perfil estimulante); recuperación del hígado; normalización del sueño.	DLPA and NAC from Day 1. 5-MTHF from Day 1. Liver support stack concurrently. Exercise starting in Week 1, even if brief. The endorphin and dopamine architectures mirror the fentanyl and cocaine guides respectively — apply the appropriate one based on the primary kratom profile.
Pain Management Track (Weeks 1–12+, ongoing)	Specialist in pain management; physiotherapist; buprenorphine if experiencing pain and opioid use disorder.	Non-opioid chronic pain management; physical therapy; buprenorphine optimisation for pain.	The person using kratom for chronic pain has not had the pain addressed—they have managed it with kratom. Stopping without a concurrent pain plan will fail. This track runs alongside recovery indefinitely.

Long Game (Months 3+)	Natural endorphin and dopamine architectures; pain management sustainability; service.	Genuine endorphin system functioning; pain managed without opioid-class substances; a life beyond kratom management.	The person who has rebuilt the endorphin system through exercise, social connections, and finding purpose in life—AND who has a proper pain management strategy—has eliminated both the physical and psychological reasons for wanting to use kratom again. This is the long-term solution.

Section 8 — The Medical Context: The Regulatory Limbo

Kratom's legal and regulatory status creates specific challenges that no other substance in this guide series faces:

- Kratom is federally legal in the US (as of 2026): the DEA withdrew its proposal to schedule it after significant public opposition in 2016. Kratom is not a controlled substance at the federal level, but it is banned in Alabama, Arkansas, Indiana, Rhode Island, Vermont, and Wisconsin, as well as in several municipalities.
- Kratom is not approved by the FDA for any medical use. The FDA has issued several warnings regarding kratom products and has tried to remove them from the market using import alerts and enforcement measures.
- The Kratom Consumer Protection Act (KCPA): is model legislation developed by the American Kratom Association that would regulate kratom for quality, labeling, and age restrictions without banning it. It has been passed in several states. It provides a regulatory framework for harm reduction within legal use.
- Because kratom is not included in standard drug screenings and is not a controlled substance, the patient's medical records may not accurately reflect their kratom use history. It is essential to disclose this information to all treating physicians.
- The Having the Physician Conversation: 'I have a kratom use disorder characterized by dependence on mitragynine and 7-hydroxymitragynine — partial mu-opioid receptor agonists. I am experiencing opioid-type withdrawal. I have been researching buprenorphine-assisted management for kratom withdrawal and I am requesting evaluation for this approach.' This framing requires the physician to engage with the clinical picture, not dismiss it.

A Final Word

THE CENTURION — The plant that was supposed to be the solution, and then became the problem.

Many people reading this guide came to kratom with the best possible reasons. They were getting off opioids without a prescriber. They were managing pain that the medical system had either undertreated or overtreated. They were finding energy for a demanding life without the criminal implications of stimulants. They were doing harm reduction, imperfectly, with what was available.

The compendium's C-37 principle — every substance preference is a signal — applies here with specific compassion. The kratom preference was a signal: a pain system that needed management, an energy system that needed support, an opioid-receptor-mediated relief from something that genuinely hurt. The signal was accurate. The solution was temporary and developed its own problem.

Recovery from kratom is not a moral correction. It is finding what the plant was providing — pain relief, energy, endorphin-mediated warmth — through means that do not themselves require management. The endorphin system the body was given can be rebuilt to provide the pain buffer that kratom was pharmacologically supplying. The dopamine architecture can provide the energy and focus that low-dose kratom was adrenergically delivering.

The opioid MAT framework, which the clinical system largely reserves for heroin and fentanyl users, applies to you. Buprenorphine works for kratom withdrawal. Ask for it by name. The physician who does not know about kratom's mu-opioid mechanism needs to be informed — and this guide provides the language to do that.

You were addressing a genuine issue. Recovery offers a more effective solution.

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American Kratom Association: americankratom.org | Treatment locator: findtreatment.gov | Crisis: 13 11 14

Refer to the Recovery Kernel Complete Centurion Compendium for all referenced protocols.

Crisis: call or text 988 | Emergency: 911 | SAMHSA: 1-800-662-4357